Splenic Trauma

Choice of Management

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The modern era for splenic surgery for injury began in 1892 when Riegner reported a splenectomy in a 14-year-old construction worker who fell from a height and presented with abdominal pain, distension, tachycardia, and oliguria. This report set the stage for routine splenectomy, which was performed for all splenic injury in the next two generations. Despite early reports by Pearce and by Morris and Bullock that splenectomy in animals caused impaired defenses against infection, little challenge to routine splenectomy was made until King and Schumacker in 1952 reported a syndrome of "overwhelming postsplenectomy infection" (OPSI). Many studies have since demonstrated the importance of the spleen in preventing infections, particularly from the encapsulated organisms. Overwhelming postsplenectomy infection occurs in about 0.6% of children and 0.3% of adults. Intraoperative splenic salvage has become more popular and can be achieved safely in most patients by delivering the spleen with the pancreas to the incision, carefully repairing the spleen under direct vision, and using the many adjuncts to suture repair, including hemostatic agents and splenic wrapping. Intraoperative splenic salvage is not indicated in patients actively bleeding from other organs or in the presence of alcoholic cirrhosis. The role of splenic replantation in those patients requiring operative splenectomy needs further study but may provide significant long-term splenic function. Although nonoperative splenic salvage was first suggested more than 100 years ago by Billroth, this modality did not become popular in children until the 1960s or in adults until the latter 1980s. Patients with intrasplenic hematomas or with splenic fractures that do not extend to the hilum as judged by computed tomography usually can be observed successfully without operative intervention and without blood transfusion. Nonoperative splenic salvage is less likely with fractures that involve the splenic hilum and with the severely shattered spleen; these patients usually are treated best by early operative intervention. Following splenectomy for injury, polyvalent pneumococcal vaccine decreases the likelihood of OPSI and should be used routinely. The role of prophylactic penicillin is uncertain but the use of antibiotics for minor infectious problems is indicated after splenectomy.

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Historical Prospective

IN 1979 Roger Sherman detailed the most complete historical prospective of splenic injury.[1] From the mid 16th century through the end of the 19th century, there were several reports of 'splenectomy' that must challenge even the most credulous reviewers. The 1549 splenectomy by Zaccarelli on a 24-year-old woman with a progressively enlarging abdominal mass is thought by many to have been an oophorectomy.[2] Both of the reported 17th century 'splenectomies' were performed for an eviscerated spleen that protruded through a penetrating anterior abdominal stab wound in one patient and through a left flank stab wound in the second patient. The spleen protruding through the left flank wound first was devascularized by ligation of the exposed pedicle and then resected 3 days later.[1] All four of the reported 18th century 'splenectomies' for trauma were performed for injured spleens that eviscerated through similar penetrating abdominal wounds. At least three of these four resections were 'partial splenectomies' of that portion of spleen that was exposed while the remaining spleen was left alone. The circumstances of the first 'splenectomy' for trauma in the United States, in 1816, are similar; the spleen eviscerated after a knife was withdrawn from the abdomen and successful removal was accomplished by ligation of the vascular pedicle and detachment of the distal part.[3] Most 19th century reports of splenectomy were accomplished with the same facility because the eviscerated spleen was excised without open celiotomy. Oh that the glories of surgery could be achieved with so little effort!

How can a penetrating object simultaneously detach retroperitoneal splenic attachments, mobilize the distal pancreas, reflect the splenic flexure of the colon, and stretch the splenic artery and vein to permit spontaneous extrusion of the spleen through the abdominal wall defect once the wounding agent has been removed? After treating thousands of patients with penetrating abdominal wounds and hundreds of patients for splenic injury, I have yet to see an eviscerated spleen. When questioned about this curiosity, none of our regional trauma surgeons have witnessed this wondrous event. Personal querying of individual surgeons representing most of the major urban trauma centers in the United States yielded not a single patient presenting with a penetrating wound filled with an extra-abdominal spleen that could be excised without celiotomy.

Following penetrating wounds, evisceration of the small bowel is a frequent occurrence. Certainly our forebears would have recognized the difference between the small intestine and the spleen; lack of recognition followed by partial or total excision of the eviscerated organ without anastomosis would certainly have led to a fatal conclusion. Colon evisceration is less frequent and may, if bloodied and edematous, be difficult to recognize; partial or total excision without anastomosis, however, would lead to the same fatal outcome. The most common organ eviscerating through a penetrating wound is the omentum. Although the eviscerated omentum usually can be recognized, the presence of blood and blood clot within the leafs and venous congestion with edema due to obstruction of venous return at the site of evisceration sometimes causes the omentum to appear as a solid organ. This extruded mass may enlarge from the combination of venous congestion and contained expanding hematoma to the point where it may actually appear like a spleen. The congestion and swelling preclude return of the swollen omentum to the peritoneal cavity, as has been described in several of the above reports. Furthermore the swollen congested hemorrhagic omentum thins out at the point of exit from the peritoneal cavity, thus, appearing as a 'pedicle,' which could be easily ligated before removal of the extra-abdominal mass. The ligated pedicle, in the 20th century, is returned to the peritoneal cavity, after which a formal laparotomy is performed. The author suspects that the only difference between this 20th century description of preceliotomy omentectomy and the earlier reports of 'splenectomy' is that our forebears did not perform the formal laparotomy, which would have demonstrated an uninjured spleen in its normal anatomic location. Consequently one must view any perceived relationship between these historical 'splenectomies' and subsequent infectious complications as being spurious and completely unrelated to an asplenic state.

The modern era of splenic surgery for trauma began in 1892 when Riegner reported the first successful splenectomy for blunt injury.[4] His patient was a 14-year-old construction worker who fell from a height and hit his abdomen. He presented with the combination of abdominal pain, abdominal distension, tachycardia, and oliguria. Through a large abdominal incision, Riegner performed total splenectomy for a badly shattered spleen, part of which was lying free in the peritoneal cavity. Two major milestones were established by this procedure. First the advantage of definitive laparotomy rather than abdominal compression for refractory intra-abdominal bleeding was reinforced by the successful outcome. Second the successful outcome helped establish the role of splenectomy as a safe and effective procedure for refractory intra-abdominal bleeding. This success also helped lay to rest the 19th century concept that the spleen was necessary for life.

Splenic Function

Throughout the centuries, splenic worth has vacillated from the lofty pedicle of an essential organ for life to the lowly depths of a vestigial organ without merit. More than 2000 years ago, Aristotle suggested that the spleen had no purpose.[5] Pliny, the first-century historian, proposed that the congested spleen hinders runners.[1] Modern anatomists might wonder how Pliny knew of the spleen, let alone that it had the potential for congestion. Pliny added that the asplenic state might cause loss of humor or laughter; one can only be intrigued by the data base that led to this conclusion. The concept that the spleen produces laughter was perpetuated through the 16th century in the Babylonian Talmud.[1] Later Maimonides postulated that the spleen was responsible for the purification of blood and not laughter; presumably impure blood hinders laughter.[6] In the many years that followed, the spleen escalated in status and, by the 17th century, was considered essential for the maintenance of life. This lofty position eroded gradually as new data emerged. Malpighi performed ligation on the splenic artery in dogs who had no untoward effects; this helped demonstrate that the avascular spleen could also sustain life.[7] During the next 100 years, splenectomy was accomplished in several animal species, all of which survived the asplenic state. Pearce,[8] in 1918, reported that 25% of animals subjected to splenectomy die from peritonitis or pneumonia. He thought these deaths were unrelated to the asplenic state because he concluded that the animal survives without the spleen with 'relative impunity.' He did note, however, that the hematologic changes following splenectomy included anemia, eosinophilia, and leukocytosis, all of which were of unknown significance.

Despite this review by Pearce, little attention was directed toward the role of the spleen in the defense against infection. Morris and Bullock,[9] in 1919, were the first to document, in a controlled scientific model, the importance of the spleen in defense against infection. These authors performed splenectomy in rats that were then subjected to a Pasteurella bacterial challenge and compared to well-established responses in control eusplenic rats. These authors concluded that the spleen in rats helps fight infection and that the removal of the spleen eliminates this defensive mechanism. These authors further expressed a concern that humans who survive splenectomy may be unable to resist a subsequent infectious insult. Their warning is precise and prophetic, stating that is was probable '. . . that the human body deprived of its' spleen would show . . . increased susceptibility to infection and some of the fatalities . . . attributed to infection . . . may be due to splenectomy.'[9]

Despite these early warnings, the surgical community moved down a road of wonting splenectomy with little or no regard for splenic salvage. Many surgical leaders, including Bailey, Moynihan, and others reported on small numbers of patients with poor follow-up and concluded that the fears of increased susceptibility to infection after splenectomy were unfounded.[1,10,11] Despite the pleas by Pfeiffer,[12] in 1924, for a more careful follow-up of these patients, the same teachings persisted and the controversy disappeared for a generation. Possibly the golden era for the evolution of individual surgical giants squelched minority views, including those regarding the potential importance of splenic salvage.

Thirty years later King and Shumacker[13] revived the controversy when they reported on five children who developed severe overwhelming infection after splenectomy for congenital spherocytosis.[13] Two of these five children rapidly died from the syndrome, which has since been named overwhelming post splenectomy infection (OPSI). Stimulated by this report, other authors identified OPSI following splenectomy performed in children for other hematologic disorders.[14,15] Initially the occurrence of OPSI after trauma was not appreciated so that the infectious sequelae in these children were thought to result from the underlying disease process rather than the asplenic state.[14-16] In 1957 Smith[17] made the first report of OPSI following splenectomy for trauma. This milestone report stimulated other centers to identify the same phenomenon in injured patients so that, by 1970, most centers recognized OPSI following splenectomy for trauma in children. Consequently discussion of splenic function and infection in children led to the concept that the immune system was immature in children.

The spleen, at birth, is considered functionally and histologically immature.[18,19] The neonatal spleen has few lymphoid follicles and little or no germ centers. This raises questions about the humoral capabilities of the young spleen in response to antigenic stimulation. By puberty the spleen increases to its maximal weight and represents approximately 25% of the total-body lymphoid mass. Does this increase in size represent the full humoral capacity of the spleen or is this simply the lymphoid mass response to many different antigenic stimuli received during this phase of maturation? The splenic blood flow by puberty and in the adult is generous, averaging 25 mL per minute. This splenic blood flow, presumably, facilitates leukocyte sequestration and the production of immunoglobulins and antibodies to circulating antigens. The efficacy of splenic function is tested partially by the ability to clear particulate materials or bacteria from the blood stream. Because a portion of this clearance may be related to splenic filtration, maintenance of generous splenic blood flow and splenic mass appear necessary to sustain this function.[19,20] Several studies show that splenectomy reduces intravascular clearance of particulate matter, including antigens. This impairment can be correlated with decreased opsonic factor synthesis and decreased release of antibodies in response to specific antigen stimulation.[21] One opsonic factor is tuftsin, a peptide that is cleaved from the gamma globulin molecule, leukokinen, by a polymorphonuclearcyte (PMN)-containing enzyme. The product of this intrasplenic cleavage circulates and, when acting in concert with PMNs, enhances opsonization and phagocytosis of bacteria.[21] The asplenic state leads to a decrease in tuftsin and immunoglobulins so that their defense activities have disappeared by 8 weeks. This includes impaired alveolar macrophage phagocytosis and depressed T-cell (T-helper) activity. The greatest reduction in bacterial clearance is seen with the encapsulated gram-positive bacteria. The efficacy of bacterial clearance is proportional to splenic mass, thus making a further case for partial splenic salvage.[20] Reduced bacterial clearance, however, is not limited to the gram-positive encapsulated organisms. Ohshio and coworkers demonstrated a postsplenectomy decrease in IgG anti-lipopolysaccharide antibody complex when mice were immunized on postsplenectomy days 1, 3, and 7 as compared to mice undergoing sham laparotomy without splenectomy.[22]

The impaired immune response after splenectomy is not limited to intravascular phenomena. Because pneumococcus is an air-borne bacterium, the respiratory route of entry also has been studied. Indeed the lungs are probably the source of OPSI in some patients.[23,24] In 1989 Hebert[23] showed that splenectomy in mice leads to decreased bacterial clearance from the lungs after an aerosol challenge of known quantities of streptococcus pneumoniae type III given 2 weeks after operation. Reduced clearance in this model is associated with altered translocation of bacteria from the tracheobronchial air passages to regional lymph nodes where antimicrobial activity occurs.[25] The complexity of these interwoven but cryptic defense activities is enormous and probably involves altered phagocytosis and reduced PMN efficacy. Among many other functions, this impaired defense against an aerosol bacterial challenge is lethal and can be ameliorated by partial splenic salvage.[23,25] Mice with 50% splenectomy and 25% splenectomy had, proportionally, better clearance and better survival. The decreased clearance of particulate matter coincides with impaired ability to clear *Escherichia coli.*

The intact spleen also is important for a normal response to vaccination with polyvalent pneumococcal vaccine. Ohshio[22] showed that preoperative immunization would not prevent a decrease in antibody response when splenectomized mice were challenged 30 days after splenectomy with the same antigen. They concluded that the intact spleen is necessary to mount a recall response of the anti-lipopolysaccharide antibody complex to a postoperative antigenic stimulation. Hosea[26] and coworkers also demonstrated an impaired antibody response to pneumococcal vaccine after splenectomy. Rabbit studies demonstrated reticuloendothelial system failure to clear particulate material from the blood stream following splenectomy. This reticuloendothelial system failure was associated with a decrease in plasma fibronectin levels with or without the added insult of sepsis that was established by open appendectomy without ligature.[26]

This impaired immunologic response contributes to the development of OPSI. This syndrome is now well established in children, with an incidence about 0.5%. The clinical features are dramatic and suggest a bacteremic shower.[1,17,18] This produces constitutional symptoms that are multisystem and devastating. The gastrointestinal symptoms include nausea, vomiting, and diarrhea. The general cardiovascular symptoms include overwhelming weakness, progressing to a prostrate state. Worsening headache is associated with increasing fever leading to coma. This overwhelming syndrome may progress from onset to death within 24 hours. Late cardiovascular collapse is associated with a rising pulse rate and hypotension. Varied electrolyte abnormalities may be encountered and these include hypoglycemia, despite glucose administration and hyponatremia mimicking adrenal insufficiency. Indeed some patients have adrenal changes on postmortem examination.

The organisms responsible for OPSI include pneumococcus, which occurs in at least 50% of patients, meningococcus, *E. coli*, *Haemophilus influenza*, Staphylococcus, and Streptococcus.[27] The clinical syndrome suggesting a bacteremic shower is reinforced by the frequent identification of many bacteria on culture and sensitivity studies and the occasional bacterium seen on a peripheral blood smear. Antibiotics, even when administered early, may fail to avert the progressive nature of this syndrome, which has a mortality rate of more than 50%.

This syndrome of OPSI probably occurs also in adults.[28] I became convinced of OPSI in adults in 1977 when a former student, now an infectious disease expert, detailed the successful treatment of OPSI in a healthy 29-year-old adult 6 years after splenectomy for blunt trauma. This patient almost died from meningococcus meningitis and pneumonitis. After splenectomy, I gave the patient neither polyvalent vaccine nor advised the patient about increased risk for subsequent OPSI and the importance of early antibiotic therapy for subsequent infections. Despite similar anecdotal examples by other surgeons, the surgical community is still slow to accept OPSI in the adult population. I recall vividly the verbal abuse from a well-known fellow panel member in 1979 after encouraging enhanced efforts at splenic salvage to preclude the risk of OPSI in adult patients. Fortunately the pendulum has swung toward splenic salvage in adult patients during the past decade.

Splenic Salvage

Operative

The story of splenic salvage in Western medicine is not exemplary. Despite frequent anecdotal reports on the feasibility of splenic salvage and the late risks of splenectomy, the routine for splenic injury remained total splenectomy. In 1897 Zikoff[18] may have performed the first splenorrhaphy for trauma. In the next generation, isolated reports of splenorrhaphy appeared but received little attention. Dretzka, the first Chief of Surgery at Detroit Receiving Hospital, reported the first splenorrhaphy in a child.[1,18] This report, in 1930, clearly outlines the technical considerations necessary for successful repair. A generous incision permits full exposure to the left upper quadrant contents. When splenic bleeding is contained by a pack, the surgeon may divide the greater omentum outside the gastroepiploic vessels, mobilize the splenic flexure of the colon inferiorly, and retract the greater curvature of the stomach anteriorly and inferiorly to identify and divide the short gastric vessels. The retroperitoneal and diaphragmatic splenic attachments cán then be divided as the plane between the posterior surface of the spleen with pancreas and the retroperitoneal tissue can be dissected. The surgeon must resist the temptation to pull the partially mobilized spleen into the abdominal incision. The younger surgeon must be taught to push the pancreas anteriorly and medially, thus allowing the spleen, by necessity, to come along for the ride. Once fully delivered to the incision, attention can be directed toward the technical nuances of splenic salvage. When the spleen is bleeding actively, the surgeon may forgo complete division of all attachments before mobilization. When rapid mobilization is necessary, the potential for irreparable iatrogenic trauma in enhanced, as is the likelihood for total splenectomy. Once completely mobilized, the techniques for splenorrhaphy are similar to those used for hepatorrhaphy, but the spleen must be handled more gently. Morgenstern[29] has highlighted these technical features. Most authors prefer the use of absorbable sutures to obtain splenic hemostasis.[29-33] Gently placed simple or figure-eight sutures of 3/0 absorbable suture permits sufficient compression of the splenic parenchyma to obtain hemostasis in most instances (Fig. 1). The need for gentleness cannot be over emphasized. When two sutures are placed in close proximity, one suture may be held at the desired tension while the second suture is tied precisely. This circumvents inadvertent parenchyma tearing. The intraparenchyma splenic vessels course both parallel and perpendicular to the hilar vessels and can be controlled with fine crisscrossing sutures using, in the authors hands, 4/0 silk (Fig. 2). Bleeding from needle holes should be contained by gentle pressure for 5 to 10 minutes or the application of a pack over the splenic repair as the spleen is returned to the left upper quadrant. The pack, when kept opposed to the splenorrhaphy site while the surgeon corrects problems elsewhere, completes the hemostasis. Once good hemostasis is achieved, the spleen should no longer be mobilized.

When the spleen is too badly injured for simple splenorrhaphy, splenic salvage may be achieved by partial splenectomy, remembering that the amount of preserved splenic mass correlates with maintained resistance against infection (Fig. 2). During partial splenectomy, the intraparenchymal vessels and the margin of resection are repaired by the techniques outlined above. Preserving intact capsular coverage of resected margins with denuded parenchyma facilitates hemostasis. Hilar vessels supplying the resected portion can be clipped or tied, with the surgeon being careful to avoid injury to those hilar vessels feeding the remaining spleen. The severely pulverized segment of spleen is best treated by resectional debridement back to an area of reasonably normal splenic substance that will hold sutures.[29-33]

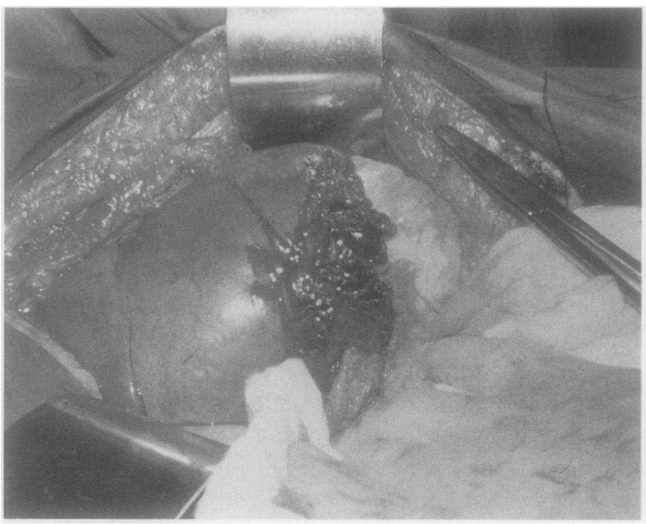

FIG. 1. After full mobilization, carefully placed and gently tied 3/0 absorbable suture splenorrhaphy provides good hemostasis in most type II splenic injuries without hilar vessel injury.

Besides splenic suture and direct pressure, several other techniques have been promulgated for intraoperative splenic salvage (Fig. 3). Electrocautery is very beneficial for minor oozing from denuded parenchyma, the splenic capsule, and small intraparenchymal vessels.[32] The ultrasonic surgical aspirator has been described for partial splenectomy.[34] Hemostatic agents containing a mixture of fibrinogen, thrombin, and collagen have been applied successfully to splenic injuries topically (Fig. 4).[35] Polyglycolic acid mesh can be wrapped around the badly injured spleen and then sewed to itself to maintain the splenic substance within its normal anatomic confines.[36] Delany[37] and coworkers wrap and suture this mesh to the badly injured spleen. The laser has been used for splenic salvage in dog studies.[28] Rabo and Ger[38] proposed stapling for splenorrhaphy. Using 4.8 mL and 3.5 mL staples, these authors perfected the technique in canine studies before successful clinical application. Despite the plethora of ancillary techniques used to augment the surgeon with splenic salvage, none of these techniques will replace good, careful, gentle surgical technique and mature surgical judgment.[35]

Using these techniques, the success rate of operative splenic salvage is quite good. Surgical teams, dedicated to operative splenic salvage in the adult patient, will achieve success in at least 50% of patients after both blunt and penetrating injury.[30-33] Moore and coworkers[32] achieved splenic salvage in 85 of 170 patients. The best success was achieved in those patients who required hemostasis by either electrocautery or the local application of topical hemostatic agents; these patients had lesser injuries and none required reoperation. The need for sutures or debridement for hemostasis was associated with a 2.5% incidence of postoperative rebleeding requiring reoperation. The patients with more severe splenic injury requiring a partial splenectomy had a 10% incidence of significant rebleeding requiring reoperation. Feliciano and coworkers[31] reported a similar success with operative splenic salvage. The national incidence of operative splenic salvage will depend on the emphasis and dedication by each surgical group. Consequently the success rate of splenic salvage varies from as low as 5% in adults to as high as 85% in children.

Splenic salvage has spread into the arena of surgery for pancreatic injury.[32] Those patients with type II pancreatic injury thought to involve the main pancreatic duct in the body or tail of the pancreas traditionally have been treated by a distal pancreatectomy with splenectomy. This operation can be performed without splenectomy.[32] Careful mobilization of the pancreas near the site of injury permits identification of the splenic artery and vein, which are ligated and divided as the pancreas is transected. The pancreas then is mobilized toward the spleen, with the surgeon being careful to identify individually the terminal branches of the splenic artery and vein to achieve he-

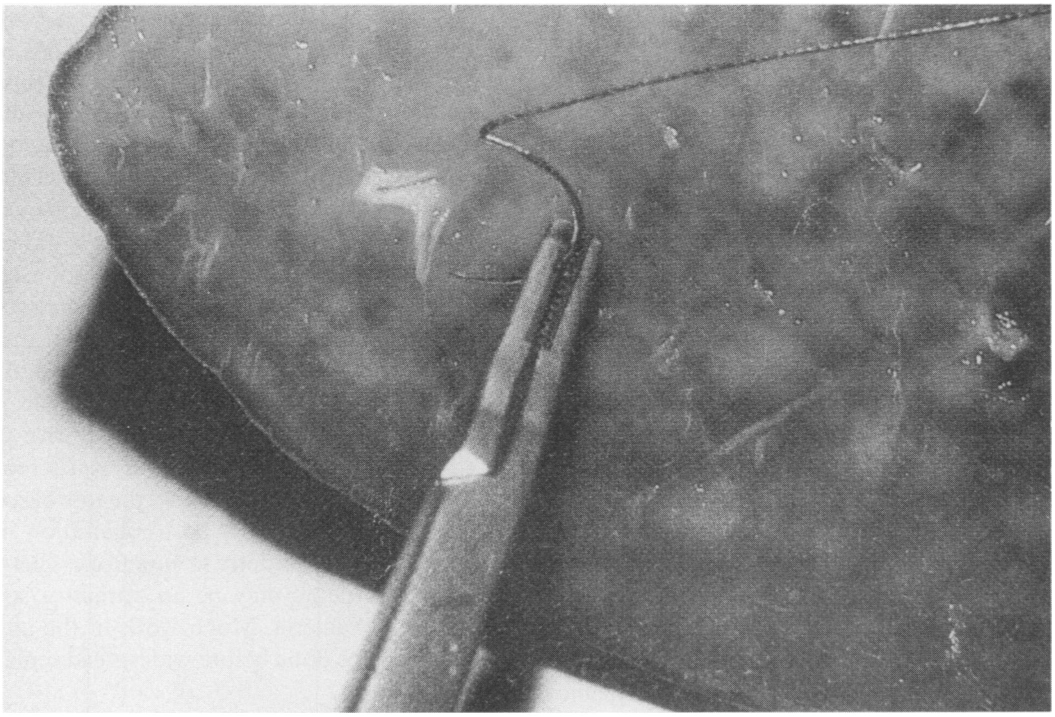

FIG. 2. Intraparenchyma vessel sutured with fine silk criss-cross suture technique provides good hemostasis to most hilar vessels.

mostasis within the splenic hilum. When feasible, the splenic artery may be preserved. The retroperitoneal and diaphragmatic attachments of the spleen are left intact, as are the short gastric vessels. The extra time needed for this operation would be unwarranted in a patient with active bleeding when pancreatectomy with splenectomy is the quickest technique for rapid hemostasis. This approach to the pancreas also can be applied to patients with combined pancreatic and splenic injury when the splenic injury is minor and can be made hemostatic by means of electrocoagulation or the topical application of hemostatic agents without requiring full mobilization from the adjacent tissues.

Some patients require splenectomy.[39] The class IV

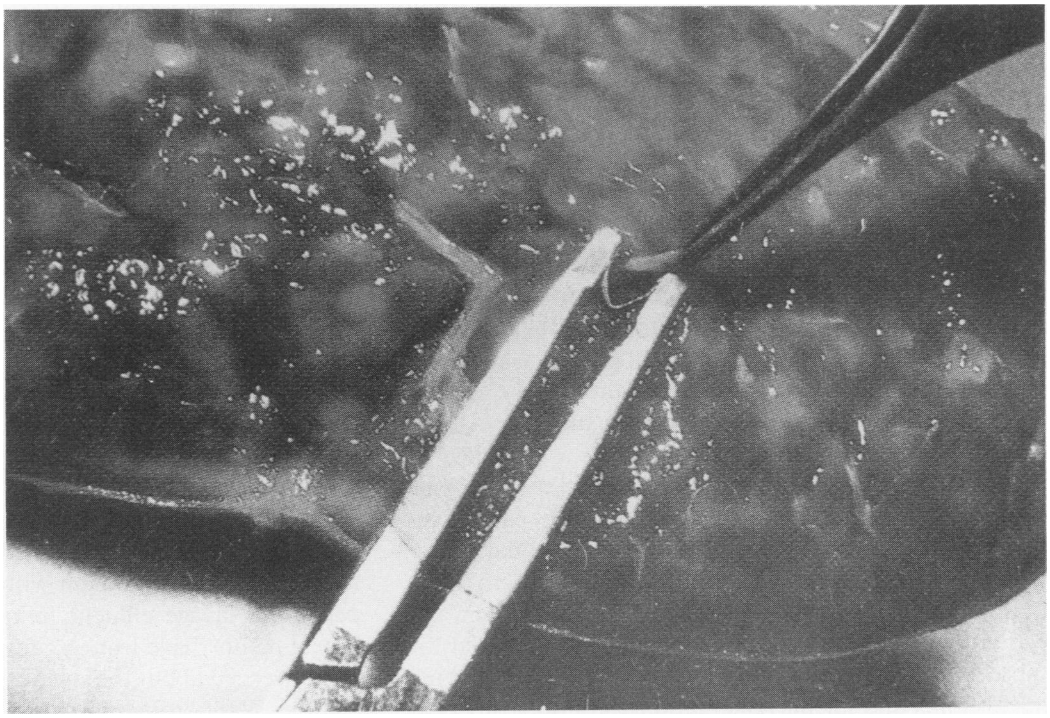

FIG. 3. Hilar vessels near hemisplenectomy margin are held by fine, smooth pick-ups before occlusion by silver clip.

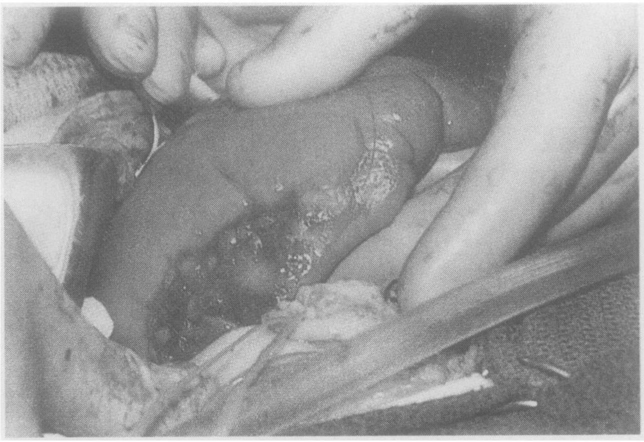

FIG. 4. Topical hemostatic agent is applied to hemisplenectomy margin to contain oozing.

splenic injuries with shattered, torn parenchyma or avulsed segments of crushed parenchyma rarely are salvaged. Patients with portal hypertension and increased bleeding from all splenic attachments uniformly require splenectomy. Patients with associated splenic disease, such as splenitis, infectious mononucleosis, malaria, lymphoma, or other intrinsic abnormalities seldom have successful splenic salvage. Finally the extra time required for splenic salvage in patients with active bleeding from the spleen and elsewhere is unwarranted. The mature surgeon intuitively recognizes when the threat of immediate hemorrhage outweighs the threat of remote OPSI. Even after splenectomy, splenic function may be preserved by autotransplantation.[32] This is accomplished by cutting the excised spleen into small fragments measuring no more than 2 mm in largest dimension. These multiple, small fragments representing at least 50% of spleen mass are placed within omental pockets as the omentum is folded on itself and held in position with through-and-through absorbable sutures. The end product looks like a splenic omelet. Moore and coworkers[32] reported on 43 patients treated by immediate replantation.

The natural history of the splenic replanted tissue includes early extensive coagulative necrosis followed by later regeneration with the ultimate return of splenic function being delayed for 5 weeks to 6 months.[40] In 1988 Rhodes and coworkers[40] described a patient who developed an omental abscess following the splenic omelet procedure after a distal pancreatectomy and splenectomy for pancreatic pseudocyst. The patient was returned to the operating room on day 11, at which time the abscessed omentum with replant was excised and the patient recovered. He suggested that the period of coagulative necrosis may be a time for increased susceptibility to infection and that this procedure should be avoided in any patient with peritoneal soilage or with the potential for subsequent sepsis from associated conditions. Following splenectomy and splenic replantation, the author observed an unusual infectiouslike response. A 56-year-old woman did well during the first postoperative week, although adynamic ileus delayed the onset of oral nutrition. This resolved and the patient was about to go home on day 8 when she became febrile (temperature, 104°F), very ill, and jaundiced (bilirubin level, 13.7 mg/dL). She was restarted on intravenous fluids with antibiotics and slowly recovered over the next 7 days and was discharged. The reason for this bizarre response remains cryptic as it was too severe for an infectious process, which never pointed, and too early for a blood transfusion reaction. We attribute this response to the systemic and hepatic effects of necrosis and lysis of the replanted spleen fragments. These anecdotal observations may mirror the 10% incidence of multiple-organ failure or sepsis reported by Moore and coworkers.[32] Possibly the loss of splenic blood filtration during the early postreplantation interval precludes normal splenic defense function, whereas necrotic splenic fragments may be an attractive source of nourishment for bacteria. Much work in the animal laboratory needs to be done before widespread replantation becomes routine.

Nonoperative

More than 100 years ago, Billroth suggested that the injured spleen has the ability to heal itself.[1] He described the autopsy findings of a 43-year-old patient who fell at work, sustaining both head and abdominal injuries. The patient died 5 days later and the postmortem examination showed a splenic injury with no evidence of recent bleeding. Billroth observed 'from the appearance of the *rent*, and the small quantity of blood effused, we concluded that this injury might have healed up completely.' This farsighted view received little attention because the surgical community was not ready for the nonoperative therapy of splenic injury. The concept of nonoperative treatment was delayed further when the specter of delayed splenic rupture was raised. In 1932 MacIndoe[41] reported that secondary hemorrhage from splenic injury occurs frequently and has a mortality rate comparable to that seen with primary splenic rupture. Based on this statement reflecting treatment of a single patient, a concept evolved that delayed splenic rupture occurs with 10% to 15% of splenic injuries. Because diagnostic peritoneal lavage and abdominal computed tomography (CT) scans have been used for blunt abdominal trauma, the incidence of delayed splenic rupture is less than 1%. The concept that an intrasplenic hematoma would expand and rupture several days or even weeks after injury because the lysed red cells caused a slowly expanding hyperosmolar cavity is now recognized as a product of inadequate diagnostic tests. I have seen only one patient with a negative (crystal clear) peritoneal lavage effluent followed later with intraperitoneal hemorrhage from a ruptured splenic hematoma.

Concerned about the splenic protective role against infection, Wanborough is reported to have initiated nonoperative therapy for suspected splenic injury at the Sick Children's Hospital of Toronto in the 1940s.[1] Any logical discussion of nonoperative splenic salvage necessitates a review of the initial diagnostic measures used in the acutely injured patient.[42] Controversies regarding initial diagnosis and management include (1) routine exploration *versus* observation for stab wounds, (2) the value of techniques used to define peritoneal penetration, (3) the role of exploration for penetrating gunshot wounds, (4) the value of diagnostic paracentesis (DPC) or diagnostic peritoneal lavage (DPL) for penetrating wounds, and (5) the value of DPL *versus* abdominal CT scan after blunt injury. Recognizing the many diverse views regarding these issues, the following recommendations incorporate some degree of author bias.

When the clinical workup suggests ongoing bleeding of suspected intra-abdominal origin after either blunt or penetrating injury, immediate operative intervention is mandated. Following successful hemostasis, diagnosis and therapy for other injuries can be completed in the operating room. When some doubt exists about the site of massive bleeding, partial resolution can be provided immediately by DPC performed on the lateral border of each rectus muscle at the level of the umbilicus. If this 20-second test yields no blood, the surgeon should reflect a moment before opening the abdomen to control massive hemorrhage. The more sensitive but more time consuming DPL probably will confirm that the cause of unstable vital signs lies outside the peritoneal cavity. If the effluent from DPL contains less than 100,000 red cells/mm^3, has a hematocrit level less than 1%, or permits the reading of news print through standard diameter intravenous tubing, then massive hemoperitoneum is absent.

The approach to penetrating stab wounds varies among surgeons. Those who adhere to the rule that all penetrating stab wounds require exploratory laparotomy try to identify when a wound has penetrated to reduce the incidence of negative laparotomies. Techniques popularized for confirming penetration include simple digital examination with the gloved finger, sinography through the stab wound, local exploration of the stab wound to its deepest level or to the peritoneum, and DPL assuming lack of penetration when the effluent is clear. Those who prefer selective observation of patients with penetrating stab wounds rely solely on the physical findings. Thus patients with stable vital signs and no peritoneal irritation are observed. When such a patient develops a decrease in hemoglobin without pain or tenderness, abdominal CT scan is performed in search of a splenic or liver injury.

When a patient presents with a stab wound along the left lower rib cage causing hemothorax, perforation of the left hemidiaphragm may result without any signs or symptoms. Diagnostic peritoneal lavage in this instance should be considered positive when any amount of blood is returned with the effluent because the patient probably has penetration of the diaphragm. Exploratory laparotomy and repair of the diaphragmatic injury prevents the subsequent development of a diaphragmatic hernia and permits the identification of associated splenic or hollow viscus injury. When a parasplenic hematoma is left unrecognized, the patient may return after chest tube removal with left upper quadrant fullness and medial deviation of the stomach on plain abdominal films or upper gastrointestinal series (Fig. 5). Once laparotomy is performed for the left upper quadrant mass, the diagnosis of splenic injury with parasplenic hematoma is confirmed. Splenic salvage at this time often is not successful.

When a patient presents with a penetrating gunshot wound, laparotomy is recommended. When question exists about penetration of the peritoneal cavity, DPL showing any blood in the effluent confirms penetration. A negative DPL in a patient with an abdominal gunshot wound, unfortunately, may be associated with a retroperitoneal injury. Thus any patient with positive abdominal signs or symptoms needs laparotomy, at which time an injured spleen will be identified. Therefore there is no literature on the natural history of a spleen being observed following a gunshot wound. As more surgeons observe patients with left thoracic gunshot wounds with positive double-contrast

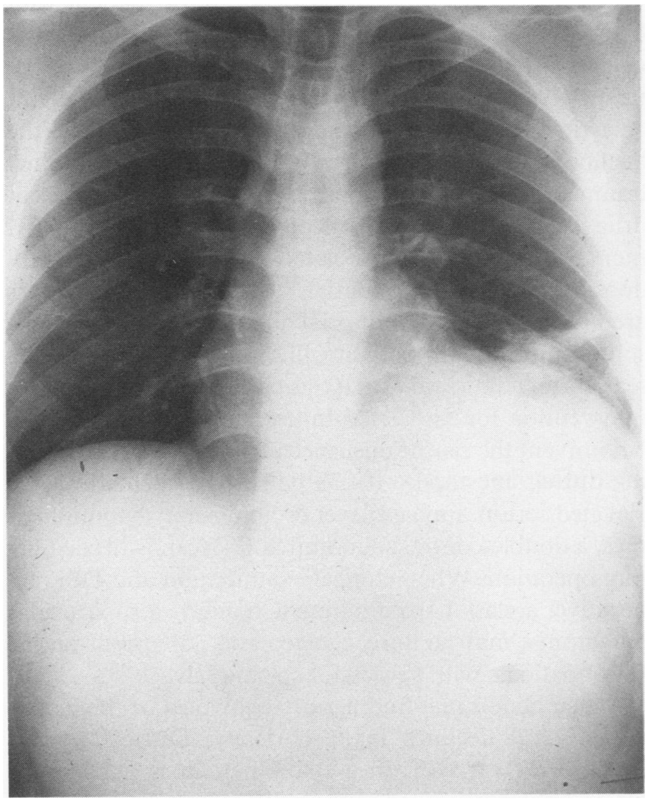

FIG. 5. Following successful tube thoracostomy therapy for a left hemothorax from a gunshot wound, this patient returned 10 days later with elevation of the left hemidiaphragm due to a ruptured spleen. Initial DPL showing small amount of bloody effluent would have confirmed diaphragmatic perforation.

abdominal CT scans, the natural history may emerge. A patient with a left lower thoracic wound with hemothorax and perisplenic blood on abdominal CT scan, despite the absence of positive physical findings, has a splenic injury. If this is observed, the natural history will be defined.

Much controversy revolves around the relative merits of abdominal CT scan *versus* DPL after blunt injury. Rather than being in competition, these two examinations are complimentary, with each having specific objectives.[42] When the patient presents with stable vital signs, a DPL, when negative, rules out hemoperitoneum. When DPL is positive, hemoperitoneum is confirmed and a judgment must be made about the presence or absence of hollow viscus injury. An elevated white cell count, high amylase content, or the presence of particulate matter or bacteria within the lavage effluent indicate hollow viscus perforation. If no obvious intraperitoneal hollow viscus injury exists, assessment is made for possible injury to the retroperitoneal organs, particularly the pancreas, duodenum, and genitourinary system. An abdominal CT scan with double contrast, including intravenous and oral contrast, provides excellent assessment of these organs. Sorkey and coworkers[42] emphasize that DPL and the double-contrast abdominal CT scan should be used in a complimentary manner to aid diagnosis and guide therapy.

Serial clinical examination of the stable patient with blunt injury frequently may lead to false-positive and false-negative conclusions. The error rate is magnified in patients with compromised mental function. The resultant delay in diagnosis and operative intervention reduces the likelihood for splenic salvage. Whenever blunt abdominal trauma occurs in association with alcohol ingestion, recent drug usage, schizophrenia or other psychic abnormality, mental retardation, or associated head injury, DPL or abdominal CT scan are essential.[42] When mental functions are uncompromised and serial abdominal exams are absolutely normal, the patient will be discharged if no other reasons for admission exit. If this patient requires operative intervention for associated injuries, DPL is indicated to circumvent the risk of unsuspected intraperitoneal bleeding during anesthesia. If this patient has hematuria, an elevated serum amylase level or abnormal abdominal x-rays, a double-contrast abdominal CT scan is needed before operation. When clinical examination and DPL are negative, a class I splenic injury, namely, a subcapsular hematoma, may go unrecognized and not appear on the list of patients with successful splenic salvage.[43]

When abdominal findings are equivocal or the hemoglobin level declines inappropriately, DPL is recommended. When DPL in a stable patient is positive for blood but negative for white cells, amylase, particulate matter, and bowel content, abdominal CT scan is warranted. When the DPL is positive for amylase, particulate matter, white cells, or bowel content, laparotomy is mandated. Whenever a patient being examined by abdominal CT develops symptoms of peritoneal tenderness or becomes unstable, immediate resuscitation and cancellation of the examination in favor of immediate operation is mandatory. Successful surgical intervention of stable patients with potentially lethal injuries is highly successful; surgical intervention of unstable patients compromises this surgical success. Thus the unstable patient should never have abdominal CT scan.

The success of nonoperative treatment of splenic injury in children has been so successful that many pediatric surgeons have abandoned DPL in favor of abdominal CT scan.[30] This approach may be, in part, motivated by the desire to avoid unnecessary laparotomy in children with positive DPL. Rather than discarding a good test such as DPL, the pediatric surgeon might modify the criteria for laparotomy based on DPL findings. When the child has a positive DPL for blood only, he or she could be watched using the same criteria for those diagnosed to have splenic injury on the CT scan. A negative DPL would, in many patients, preclude the need for an abdominal CT scan, whereas a DPL positive for bowel content or amylase would direct early operative intervention when associated splenic injury would be identified and salvaged. The disadvantage of early DPL is that not all the lavage infusate exits by gravity, so a portion of the intraperitoneal fluid seen on subsequent CT scan would be electrolyte solution and not blood. This factor should not interfere with successful nonoperative management of splenic injury. The earlier diagnosis of intraperitoneal hollow viscus injury by DPL may become more relevant with increased compliance with new laws regarding automobile back seat restraints. The incidence of hollow viscus injury in children is higher with the back seat lap restraints and these injuries are not diagnosed best by abdominal CT scan.[44]

Although calculated observation of suspected splenic injury was practiced sporadically during the 1960s, this mode of therapy was unpopular in adults until the 1980s. Most surgical teams observing suspected splenic injury in the 1970s based the diagnosis on liver-spleen radionuclide scans or arteriography. Both exams have a significant false-positive rate when compared with computed tomography.[45] Consequently one may question the published success rate of nonoperative splenic salvage in the 1970s. During the 1980s, computed tomography became established as the standard for diagnosis of splenic injury.

Even the CT scan has limitations. Buntain and Gould identified two patients with unrecognized intrasplenic hematoma diagnosed during operation performed for other reasons.[30,43] These authors offered an excellent classification of splenic injury (Table 1). Class I injuries include subcapsular or intraparenchymal hematomas without capsular disruption or parasplenic blood. Class II injuries include open tears or fractures that involve the parenchyma but do not extend into the splenic hilum. These patients have free intraperitoneal blood, which may spread

TABLE 1. *Severity of Splenic Injury*

Type	Signs
Type I	Subcapsular or intraparenchymal hematomas without capsular disruption
Type II	Open parenchymal tears or fractures that do not extend into the hilum
Type III	Large open fractures extending into the hilum
Type IV	Shattered or pulpified spleen with hilar disruption or avulsed fragments

beyond the left upper quadrant. Class III injuries extend into the hilar structures and cause more hemoperitoneum. Class IV injuries include the shattered or pulpified spleen, which may be avulsed partially from its hilar attachments. Based on CT, these authors further subdivided splenic injuries according to associated organ injuries, with subtype A patients having no extrasplenic injuries, type B_1 patients having associated solid viscus injury, and type B_2 patients having associated hollow viscus injury. Buntain and associates[43] reported that the class of splenic injury correlated significantly with the injury severity score but not with the trauma score. Successful nonoperative therapy was achieved in all class I injuries, 60% of class II injuries, 7% of class III injuries, and none of the class IV injuries. Rescinitti[33] modified this system for estimating the severity of splenic injury. The extent of splenic parenchymal injury was graded: one for a simple laceration, two for a fracture, and three for a shattered spleen. Additional points were given for hemoperitoneum confined to the left upper quadrant, hemoperitoneum remote from the spleen but not in the pelvis, and pelvic hemoperitoneum. The maximum score is six. These authors noted that nonoperative therapy was successful in all patients with scores between 1.0 and 2.5, whereas successful nonoperative treatment was possible in only 7 of 15 patients with scores between 2.5 and 5.5 points.[33]

The success of nonoperative splenic salvage after blunt trauma varies with progressive attitudinal changes among the surgeons providing emergency department coverage. These changes develop slowly, as demonstrated by the experiences of the William Beaumont Hospital in Royal Oak, Michigan. Some surgeons introduced nonoperative splenic salvage in 1978.[46] Opposition among colleagues was fierce and acceptance slow. Resistance lessened after 5 years, when nonoperative splenic salvage was achieved in 30% of injured patients and none of the observed patients required late splenectomy.[46] From 1984 through 1989, the success of nonoperative splenic salvage increased to almost 70% as 34 or 52 patients have been spared an unnecessary operation.[47] More importantly only 1 of the 18 patients requiring operation had isolated splenic injury and this patient never received any preoperative blood transfusions; his surgeon now admits operation was premature. All patients requiring blood transfusions (range, zero to seven units) during the course of nonoperative splenic salvage had associated injuries. Furthermore laparotomy in most of the 18 patients who were not candidates for nonoperative splenic salvage was dictated by the signs and symptoms of associated intra-abdominal injuries, including the liver, diaphragm, duodenum, pancreas, and intestine. The typical clinical course is presented in the following case summary.

Case Report

A 30-year-old woman presented after a motor vehicle accident. She was a restrained driver struck on the left side. Positive findings included left lower thoracic and left upper abdominal pain and tenderness, and left knee and pubic tenderness. Roentgenograms and abdominal CT scan revealed a left seventh rib fracture, a class II splenic injury, and a pelvic fracture (Fig. 6). Treatment included intravenous crystalloids and bed rest. The initial hemoglobin of 11 g/dL decreased to a final value of 9.2 g/dL on day 2. Diet was initiated on day 3 and and patient resumed a regular diet by day 5. Discharge home was delayed until day 9 for orthopaedic considerations. Repeat abdominal CT scan at 5 weeks showed splenic healing (Fig. 7).

Another patient had successful nonoperative therapy of combined type II spleen and type II liver injuries, which foretells a future narrative on the nonoperative approach to blunt liver injury (Fig. 8). The 70% success rate of nonoperative splenic salvage achieved after blunt abdominal trauma in adults is surpassed by recent pediatric experience. Wesson reports that 85% of children with blunt splenic injury can be managed successfully without operation.[48] The guidelines for nonoperative splenic salvage are emerging. The stable patient without suspected intraperitoneal hollow viscus injury is a candidate for CT. Some degree of abdominal tenderness is common but this tenderness typically is much less than one sees with hollow

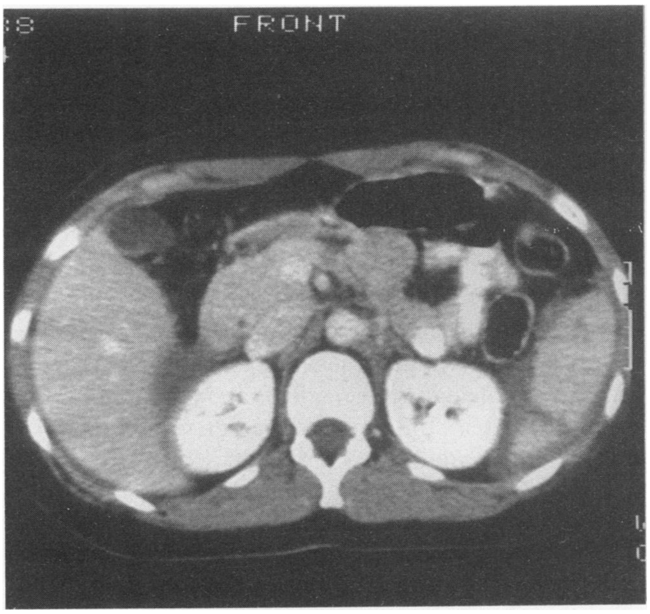

FIG. 6. Abdominal CT scan of this 30-year-old woman shows a type II splenic transection that was successfully treated nonoperatively (courtesy of Dr. Robert Lucas, Royal Oak, MI).

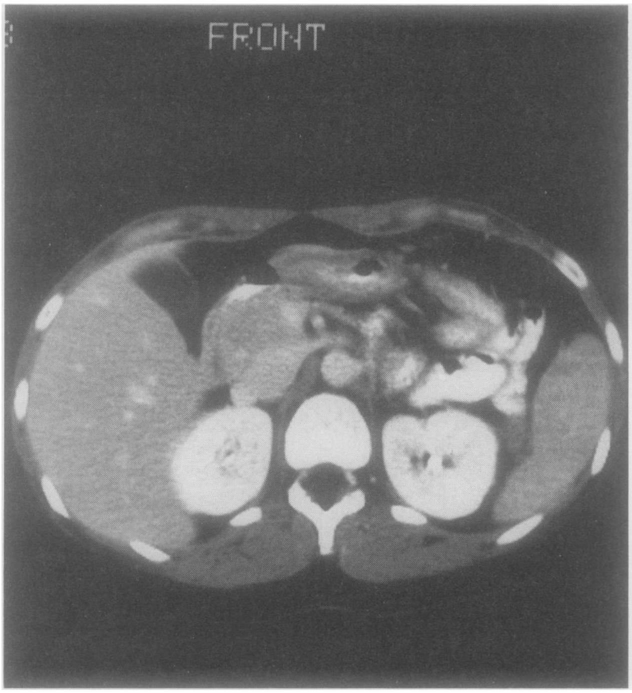

FIG. 7. Six weeks after splenic rupture, abdominal CT scan shows complete healing (Courtesy of Dr. Robert Lucas, Royal Oak, MI).

viscus perforation. When tenderness worsens during observation, exploration is indicated to preclude hollow viscus injury. Often the hemoglobin decreases and can be treated without transfusion when the hemoglobin remains more than 8 g/dL. Transfusion of two units of blood during the initial 48 hours to maintain the hemoglobin to more than 8 g/dL is compatible with successful nonoperative salvage. A further decrease in hemoglobin requires operative intervention unless the progressive anemia results from other injuries, especially fractures. The observing surgeon must be willing to reverse the choice of therapy whenever vital signs become unstable, more than two blood transfusions for splenic injury are needed, or when hollow viscus or retroperitoneal injury are suspected.

Critics of nonoperative splenic salvage in the adult patient emphasize the potential of overlooked hollow viscus perforation and the low incidence of operative splenic salvage in patients requiring late operative intervention.[49] Proponents for nonoperative splenic salvage suggest that hollow viscus perforation leads to early dramatic peritoneal signs in mentally alert patients and that nonoperative splenic salvage is contraindicated when one or more factors impairs mental competence. These proponents also rationalize that most patients who have successful splenorrhaphy could have been successfully observed nonoperatively. Probably there is a significant overlap so that the numbers of patients who can have successful splenorrhaphy but cannot be successfully observed is quite small. The threat of an overlooked diaphragmatic rupture should be small because most such injuries are apparent on plain films, whereas the remainder should be diagnosed by CT.[49]

Splenic Wound Healing

Despite the phenomenal increase in splenic salvage by either operative splenorrhaphy or nonoperative observation,[18] few studies address the efficacy of splenic wound healing. Previous studies have suggested that patients treated by splenorrhaphy or by nonoperative observation need bed rest for 1 week and should avoid contact recreation for 6 months. Dulchavsky and coworkers[18] studied the efficacy of splenic wound healing in control canine and porcine models. They created multiple identical class II lacerations extending 5 cm in length and 1 cm in depth. Wound breaking strength (WBS) was monitored following splenorrhaphy and healing by second intent after temporary pack compression for hemostasis. These injuries did not extend into the splenic hilum. The WBS after splenorrhaphy equaled WBS of uninjured splenic tissue by 3 weeks in both these animal models. The WBS after healing by second intent equaled the WBS of uninjured splenic tissue by 6 weeks in both species (Fig. 9). The increase in WBS paralleled extensive capsular fibrosis and an exuberant fibrotic reaction at the splenorrhaphy site and along the intrasplenic septa. When no intrasplenic defects exist after either operative or nonoperative splenic salvage, return to normal activities, including contact sports, should be safe by 6 weeks.[18]

Not all splenic injuries show complete resolution on CT scan within 6 weeks after injury. Smith (personal communication) observed a 39-year-old motorcyclist after the diagnosis of class II splenic injury was made by CT scan (Fig. 10). During the first 48 hours, the patient received two transfusions for correction of anemia of 6.8 g/dL. When the hemoglobin slowly decreased to 7.3 g by

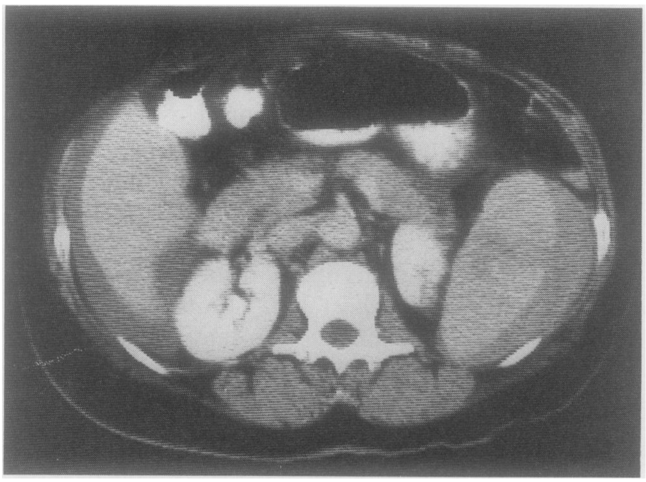

FIG. 8. Patient with type II splenic and type II liver injury (not shown) with parenchyma disruption and capsular tear, giving hemoperitoneum treated without operation. Total hospitalization was 4 days.

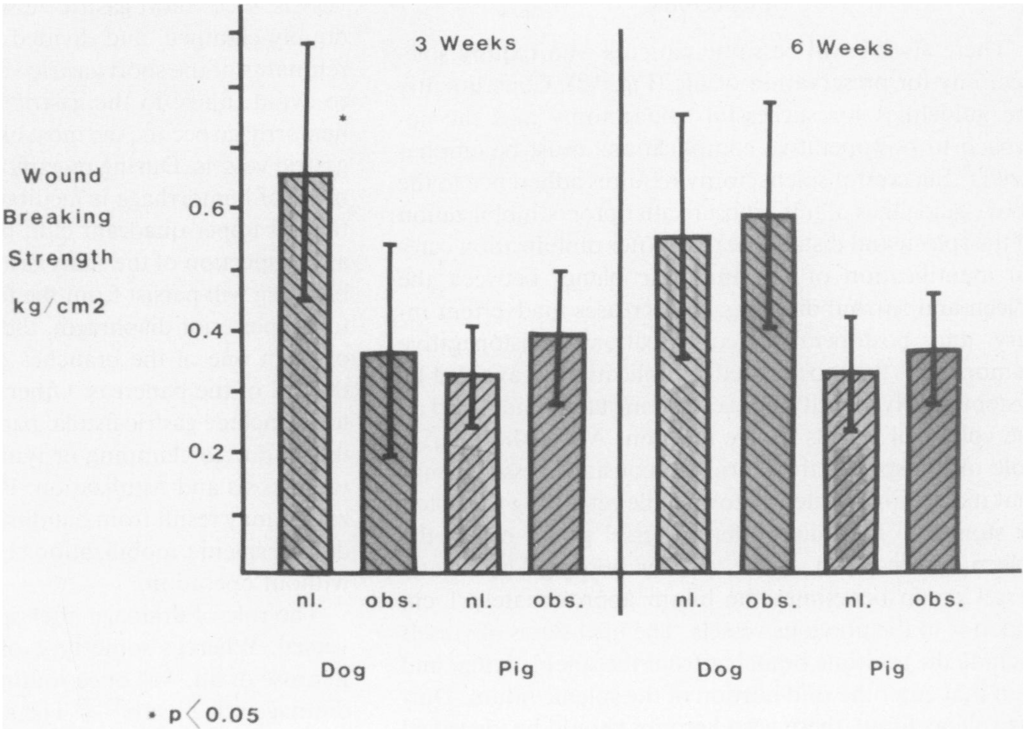

FIG. 9. Splenic wound-breaking strength recovers quickly after both splenorrhaphy and healing by second intent (obs) in canine and porcine models.

day 6, two additional blood transfusions were given. Shortly thereafter the adynamic ileus resolved and the patient was discharged on day 9. Follow-up abdominal CT scan 6 weeks later showed complete resolution of the hemoperitoneum but persistence of an intrasplenic hematoma (Fig. 11). No data on the potential for subsequent splenic rupture in this patient is available. Pending such data, the recommendation would be for continued avoidance of contact sports until a subsequent abdominal CT scan showed complete resolution of the intrasplenic hematoma.

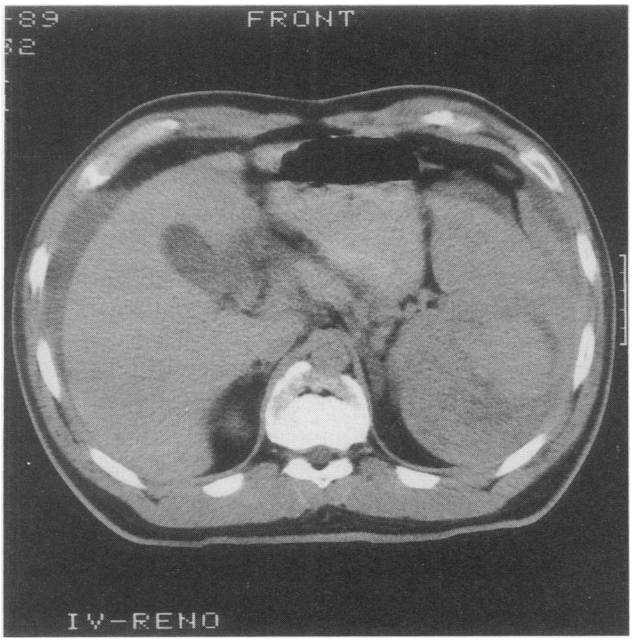

FIG. 10. Thirty-nine-year-old motor cyclist with type II splenic injury treated without operation (Courtesy of Dr. Randall Smith, Temple, TX).

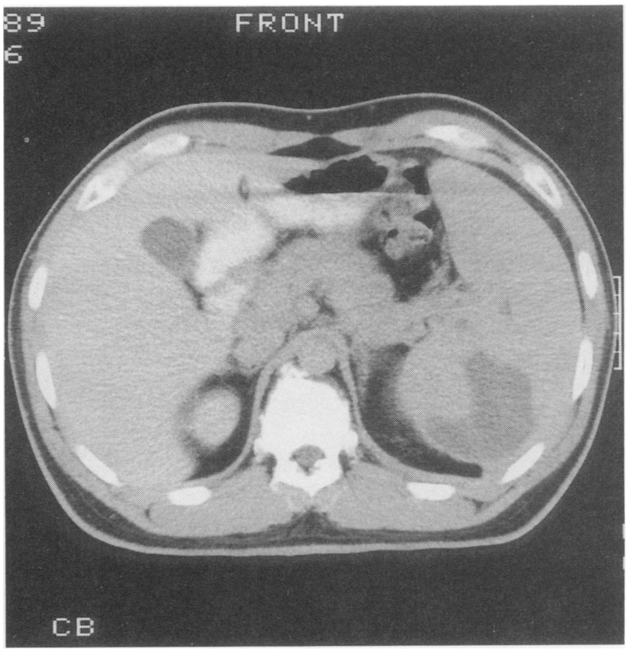

FIG. 11. Six weeks after nonoperative therapy for type II splenic injury (Fig. 10), residual intraparenchymal hematoma remains. Avoidance of contact sports is deferred until a repeat scan in 6 weeks. (Courtesy of Dr. Randall Smith, Temple, TX).

Splenectomy

There always will be some patients who require splenectomy for preservation of life (Fig. 12). Consequently the guidelines for successful splenectomy and the approach to postoperative complications must be emphasized.[29] Successful splenectomy requires adherence to the above guidelines of full exposure after proper mobilization of the spleen and distal pancreas. After mobilization careful identification of the anatomic planes between the spleen and surrounding organs decreases inadvertent injury and postoperative complications. Postoperative hemorrhage, the most feared complication, is avoided by systematically identifying each splenic attachment and all the collateral vessels before division. Along the inferior pole of the spleen, there are two constant vessel groups that may be inadvertently torn while retracting the colon or stomach. The most inferior vessel group enters the spleen within 1 cm of the inferior border. The second vessel group penetrates the hilum approximately 1 cm superior to the previous vessels. The next series of vessels include the multiple branches from the splenic artery and vein that enter the mid portion of the splenic hilum. During splenectomy, these vessel groups should be identified carefully and clamped while taking care to avoid injury to the tail of the pancreas. If active bleeding impairs full visualization, the tip of the pancreas can be palpated, thereby permitting safe division of these vessel groups without pancreatic injury. Finally care must be taken to avoid injury to the short gastric vessels. Usually two vessel groups course from the superior splenic hilum to the most proximal portion of the greater curvature of the stomach. These vessels are torn easily when the cardioesophageal junction is pulled anteriorly or medially. With careful division of the proximal branches of the left gastroepiploic vessels, these short gastric vessels can be identified easily, doubly clamped, and divided. When ligating the gastric remnants of the short gastric vessels, care should be taken to avoid injury to the gastric wall. When postoperative hemorrhage occurs, the most likely source will be the short gastric vessels. During re-exploration, successful containment of hemorrhage is facilitated by rapid evacuation of the left upper quadrant clot, packing of the splenic bed, and inspection of the short gastric vessels. Less frequently bleeding will persist from the former splenic attachments to the posterior diaphragm, the retroperitoneal structures, or from one of the branches of the splenic vessels near the tail of the pancreas. Other complications of splenectomy include gastric fistula, pancreatic fistula, and colonic fistula due to clamping or tying these organs. This leads to necrosis and fistulization. Pancreatitis without fistulization may result from handling the pancreas too roughly during splenic mobilization. Usually this can be treated without operation.

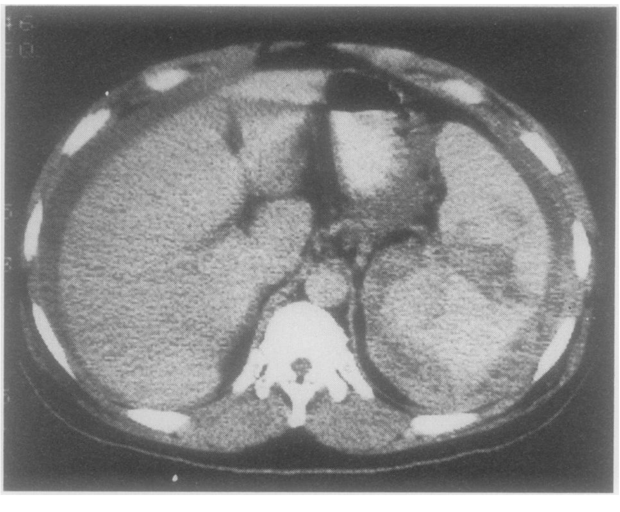

FIG. 12. After 72 hours of nonoperative therapy, this 32-year-old victim of blunt injury developed increased abdominal pain and new bleeding from this type III splenic injury extending through the hilar structures, necessitating laparotomy and splenectomy.

The role of drainage after splenectomy is also controversial. Whereas some type of drainage, usually with a Penrose drain, was once routine, most surgeons now use drainage selectively.[29-33] The author recommends drainage only for patients with associated colon injury, massive contamination from gastric or small bowel injury, or when some questions exist about the efficacy of hemostasis. In this instance, a soft rubber is brought out a 1-inch separate stab wound and enclosed within a colostomy bag to preclude stagnation of exuded fluid on saturated dressings surrounding the drain. Once drainage has ceased or decreased to less than 50 mL per 24 hours, the drain is removed. Usually this occurs by the second postoperative day before significant retrograde movement of bacteria from the skin to the subphrenic space occurs. Splenectomy also causes hematologic changes, especially with the platelets. A routine postsplenectomy thrombocytosis peaks at more than one million/mm^3 by the 10th day. This thrombocytosis is not an indication for anticoagulation because these platelets are old and do not promote clotting.

After successful splenectomy and postoperative management, the surgeon's responsibility for patient care does not end. The patient must be forewarned of the potential for OPSI and the need for prophylaxis against subsequent infection.[27,43,50] The polyvalent pneumococcal vaccines provide some degree of protection. When there is some question about patient compliance within postdischarge visits, this vaccine should be administered before discharge. Alternatively the vaccine can be administered during the follow-up office or clinic visits. The value of a booster injection with the polyvalent vaccine has not been determined yet. Powell and coworkers[27] demonstrated that the combination of polyvalent vaccine plus penicillin therapy would provide complete protection or survival after an intraperitoneal injection of *Streptococcus pneu-*

moniae in rodents. In contrast the polyvalent vaccine alone resulted in only a 25% survival rate compared to only a 5% survival rate in animals that were protected with neither the polyvalent vaccine nor the penicillin. The protection afforded by the combination of vaccine and penicillin was complete, regardless of whether the penicillin was given 3 days before or 1 day after the pneumococcal challenge. Zerrabi[50] supports the use of penicillin prophylaxis, emphasizing that the likelihood for OPSI in patients receiving prophylactic penicillin is very rare. This report must be put into perspective, however, because the incidence of OPSI in patients not receiving penicillin prophylaxis is also very uncommon.

Unfortunately the value of the polyvalent pneumococcal vaccine may be blunted by the multitude of organisms causing OPSI because many bacteria are not encapsulated or are not included in the spectrum covered by the polyvalent vaccine.[27] Furthermore some studies suggest that the efficacy of the polyvalent vaccine depends on the presence of splenic tissue.[26] Consequently the asplenic state would impair effective synthesis of antigens against the pneumococcal species. Amid these uncertainties, a reasonable course of action calls for the administration of the polyvalent vaccine before discharge in those patients thought to be noncompliant with office visits or following full recovery from the operative insult in patients seen in the office. In addition to the polyvalent vaccine, the patient should be informed of the potential for impaired immunologic defenses and be advised to communicate early with his treating physician for all infectious problems. Institution of early penicillin therapy or some similar antibiotic coverage for infectious problems should provide added protection against OPSI. Pending subsequent studies, the use of prophylactic antibiotic therapy in patients without infection is not recommended.

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